Genitourinary Medicine

Edited for the Medical Society for the Study of Venereal Diseases

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Notices

Organisers of meetings who wish to insert notices should send details to the editor (address on the inside front cover) at least eight months before the date of the meeting or six months before the closing date for application.

Courses on the acquired immune deficiency syndrome (AIDS)

The Royal College of Physicians of London is organising courses to train general physicians who will be concerned in the care of patients with AIDS. Each course will last for one week (Mondays to Fridays); mornings will be spent at the College and afternoons at one of four hospitals with major AIDS centres in London (St George's, St Mary's, St Stephen's, and the Middlesex). Numbers on each course will be limited to 20, with groups of five attending each hospital. The fee will be £90, and buffet lunch at the college each day and coffee or tea are included.

Starting dates and closing dates for applications are as follows:

Week starting 1988	*Closing date for applications*
14 March	12 February
5 September	26 July
21 November	10 October

For further details and application form, please contact: The Assistant Registrar, Royal College of Physicians, 11 St Andrew's Place, Regent's Park, London NW1 4LE (tel: 01 935 1174).

Fifth Zagazig international conference of dermatology and venereology

The fifth Zagazig international conference of dermatology and venereology will be held on 6 to 10 February 1988 in Cairo, Egypt.

For further information please contact Professor M Amer, Chairman, Dermatology and Venereology Department, Faculty of Medicine, Zagazig, Egypt.

Anglo-Scandinavian conference on sexually transmitted diseases

The Anglo-Scandinavian conference on sexually transmitted diseases will be held on 11 to 13 May 1988 at the Royal Society of Medicine, London.

For information please contact the conference secretariat (Miss Barbara Komoniewska) at the Royal Society of Medicine, 1 Wimpole Street, London W1 (Tel: 01 408 2119 ext: 301).

First conference of the European Society for Chlamydia Research

The first conference of the European Society for Chlamydia Research will be held on 30 May to 1 June 1988 in Bologna, Italy. The main topics will include: epidemiology and preventive measures against chlamydial infections (with *C trachomatis* and *C psittaci*) in Europe, biology, clinical manifestations and treatment, immunology and interaction between host and parasite, diagnostic procedures, chlamydial genetics, and vaccine development.

Please contact Dr Roberto Cevenini, Institute of Microbiology, University of Bologna, S Orsola University Hospital, 9 via Massarenti, 40138 Bologna, Italy (tel: 051-341652/302435).

34th General assembly of the International Union against the Venereal Diseases and Treponematoses (IUVDT)

The 34th general assembly of the IUVDT will be held on 3–4 June 1988 at the Palais des Festivals de Cannes, Cannes, France.

For information please contact Dr MA Waugh, Secretary General of the IUVDT, The General Infirmary at Leeds, Great George Street, Leeds LS1 3EX. (Tel: 0532 432799).

Spring meeting of the Medical Society for the Study of Venereal Diseases (MSSVD)

The spring meeting of the MSSVD will be held on 30 June to 3 July 1988 at Cambridge, England.

For information contact the local organiser, Dr MR FitzGerald, Addenbrooke's Hospital, Cambridge, CB2 2QQ or the travel agent, Mr Peter Davidson, Horncastle Travel Limited, 10 Market Street, Newcastle-upon-Tyne, NE1 6JF.

Australian and New Zealand conference on sexually transmitted diseases

An Australian and New Zealand conference on sexually transmitted diseases will be held on 25 to 27 August 1988 at the University of Melbourne, Melbourne, Victoria, Australia.

For further information please contact: The Manager, National Australia Bank Ltd Travel Groups/Incentives, 271 Collins Street, Melbourne, Victoria, Australia 3000.

Institut Alfred Fournier Prix de l'Association des Anciens Élèves et Compagnons, 1988.

Deux prix d'un montant de fr 15 000 chacun destinés à récompenser un travail original ou un ensemble de travaux, dans le domaine des maladies transmises par voie sexuelle (MST),
—l'un en sciences fondamentales
—l'autre concernant le ou les sujets suivants: Épidémiologie—Biologie—Clinique—Thérapeutique

Les candidats devront adresser le texte de leur travail définitif, dactylographié et rédigé en français, présenté sous forme d'une publication, en six exemplaires, avant le 15 Septembre 1988.

La remise solennelle des Prix 1988 se fera lors de l'Assemblée Générale de l'Association des Anciens Élèves et Compagnons d'Alfred Fournier, en Novembre 1988,

Pour toute demande de renseignements et envoi de candidature, s'adresser au:
Secrétariat de l'Association, Institut Alfred Fournier, 25 Boulevard Saint-Jacques, 75680 PARIS CEDEX 14, (Tel: (1) 45 65 27 77).

List of current publications

Selected abstracts and titles from recent reports published worldwide are arranged in the following sections:

Syphilis and other treponematoses

Acquired syphilis in a patient with late congenital syphilis

K PAVITHRAN (Kottayam, India). *Sex Transm Dis* 1987;**14**:119–21.

Neurological relapse after benzathine penicillin therapy for secondary syphilis in a patient with HIV infection

CD BERRY, TM HOOTON, AC COLLIER, SA LUKEHART (Seattle, USA). *N Engl J Med* 1987;**316**:1587–9.

The single case presented is of a 26 year old homosexual man who had been an intravenous heroin user. Serological tests for syphilis gave negative results until he presented with a rash typical of secondary syphilis. He had no lymphadenopathy or other signs of early syphilis. Syphilis serology tests gave positive results, including a titre of 1/256 in the Venereal Disease Research Laboratory (VDRL) test. He was treated with a single dose of 2·4 MIU intramuscular benzathine penicillin, and within three months the VDRL titre had fallen to 1/16. Five months after his initial treatment he developed a right hemiparesis and homonymous hemianopia with expressive aphasia. The right pupil reacted sluggishly to direct light, but the consensual response was normal. He had no other abnormal physical signs. A head scan by computed tomography and angiography showed an extensive infarct near the left internal carotid artery. His serum VDRL titre had risen to 1/256 and the VDRL titre of the cerebrospinal fluid (CSF) was 1/4, with raised white cell count (32×10^6/l, 93% mononuclear cells), red cell count (54×10^6/l), and protein concentration (1·07 g/l). No evidence of toxoplasmosis or connective tissue disease was found. He was treated with intravenous crystalline penicillin G for 14 days. His CSF and serological results improved, but his clinical condition did not. His recent sexual partners had no evidence of syphilis (VDRL result only given).

The authors pointed out that there have been other reports of failure of treatment with penicillin. They stated that, after his apparent cure, their patient developed features typical of meningovascular syphilis, which they suggest was because treponemicidal concentrations were not achieved in the CSF. Other studies have shown benzathine penicillin to yield only low or undetectable concentrations in the CSF, and one patient has been described in whom *Treponema pallidum* was isolated from the CSF despite treatment. Data on penicillin concentrations in this patient's CSF were not included.

The authors discussed the possible role of HIV. Though the patient had antibodies to HIV at the time of the stroke, no previous result was given. The importance of HIV is therefore impossible to establish in this case. They hypothesised that if damaged cell mediated immunity, as shown by skin test anergy in this patient, affects the response to syphilis, then treponemes might be expected to proliferate and disseminate if treatment has not eradicated them. The authors did not consider that HIV infection was the direct cause of this patient's stroke. They concluded by drawing attention to the hazards of inadequately treated infection and the need for further studies into the role of lumbar puncture, the efficacy of currently recommended treatment regimens, and the possible effects of HIV infection on the progression of syphilis and its response to treatment.

R J C Gilson

Alteration in the natural history of neurosyphilis by concurrent infection with the human immunodeficiency virus

DR JOHNS, M TIERNEY, D FELSENSTEIN (Boston, USA). *N Engl J Med* 1987;**316**:1569–72.

Hair root studies in patients suffering from primary and secondary syphilis

AH van der WILLIGEN, JDR PEEREBOOM-WYNIA, JCS van der HOEK, PGH MULDER, T van JOOST, E STOLZ (Rotterdam, the Netherlands). *Acta Derm Venereol (Stockh)* 1987;**67**:250–4.

Detection of immunoglobulin M antibodies to *Treponema pallidum* in a modified enzyme-linked immunosorbent assay

F MÜLLER, M MOKOPHIDIS, H-L BORKHARDT (Hamburg, Federal Republic of Germany). *Eur J Clin Microbiol* 1987;**6**:35–9.

The hyaluronidase associated with *Treponema pallidum* facilitates treponemal dissemination

TJ FITZGERALD, LA REPESH (Duluth, USA). *Infect Immun* 1987;**55**:1023–8.

Treponema pallidum contains hyaluronidase, which is associated with its surface. Seven years ago the authors proposed that this was the cause of the attachment of *T pallidum* to cultured cells, but now wish to explore the hypothesis further using Nicholls strain *T pallidum* maintained by passage in cortisone treated rabbits' testes. Three types of serum were used in the study; normal (from healthy rabbits giving negative results to the

rapid plasma reagin test), immune (from rabbits initially infected intradermally and then repeatedly challenged), and those with antibodies to hyaluronidase (obtained by repeatedly injecting rabbits with bovine hyaluronidase). All serum samples were heated to inactivate complement.

Immobilisation of *T pallidum* was tested by incubating these three sera with fresh rabbit complement and *T pallidum* in 2·5% oxygen at 37°C. The time for 50% immobilisation was recorded. Neutralisation was tested using the three sera and *T pallidum* suspensions without complement and, after incubation at 37°C in 2·5% oxygen, injecting the mixtures intradermally into rabbits. The results showed that antibodies to hyaluronidase did not immobilise or neutralise *T pallidum*, either in vitro or in vivo, when antibodies were raised in rabbits by injecting bovine hyaluronidase before injecting *T pallidum* intradermally. Immune sera both neutralised and immobilised *T pallidum*. Attachment to fibroblasts, and to capillaries isolated from brain tissue, was tested by incubating *T pallidum* with cell suspensions and the three sera. The numbers of organisms on 50 cells were counted. Immune serum blocked attachment to cells (71% block) and capillaries (83% block). Serum with antibodies to hyaluronidase caused 52% blockage on capillaries, but none on fibroblasts.

Rabbit amnions from 27–30 day pregnancies were removed and placed in Bionique chambers. The amnions consisted of a layer of epithelium, a layer of basement membrane, and a layer of hyaluronic acid. The epithelial layer was in contact with *T pallidum* extraction medium. *T pallidum* suspensions incubated with the three test sera were placed on top of the amnions, and migration through them was assessed by sampling from the extraction medium. Both immune serum samples and those with antibodies to hyaluronidase retarded penetration. Amnions treated with ammonium hydroxide to leave only basement membrane were used similarly but in this experiment only immune sera retarded penetration. Exogenous hyaluronic acid added to these treated amnions caused retarded penetration in the presence of antibody to hyaluronidase.

Evans blue was injected intravenously before dermal inoculation of *T pallidum* to test for vessel leakage after the injection. After incubation with the three test sera, *T pallidum* suspensions were injected intradermally into rabbits' backs. Normal serum had no effect on vessel damage, immune sera reduced it slightly, and sera with antibodies to hyaluronidase caused the greatest inhibition of vessel damage.

Testicular dissemination was examined by incubating *T pallidum* with normal sera and those with antibodies to hyaluronidase. The suspension was injected into the right testis of 20 rabbits, 10 receiving the normal sera plus *T pallidum*, and 10 receiving serum with antibodies to hyaluronidase plus *T pallidum*. After 10 to 13 days the rabbits were killed, each testis extracted, and *T pallidum* present counted. In the rabbits receiving normal sera the right testis count was $6{\cdot}62 \times 10^7$ and the left $0{\cdot}11 \times 10^7$ organisms/ml. Those receiving sera with antibodies to hyaluronidase had $7{\cdot}79 \times 10^7$ on the right and $0{\cdot}02 \times 10^7$ on the left.

The authors originally postulated that *T pallidum* attached to cultured cells by its hyaluronidase interacting with the cellular hyaluronic acid. If this was true, prior incubation with antibodies to hyaluronidase should result in decreased cellular attachment. This was not the case. Cellular attachment had also been thought to be an important part of infection, but antibodies to hyaluronidase did not decrease treponemal virulence. The authors now present a speculative model: *T pallidum* reacts initially by hyaluronidase binding to cultured cell hyaluronic acid. After enzymatically degrading the surface coating of hyaluronic acid, the organism directly contacts cultured cell constituents, such as collagen, fibronectin, and laminin, and binds to them via specific ligands, which may be responsible for the characteristic end-on firm attachment. In vivo this would mean treponemal hyaluronidase acting as a spreading factor to enhance dissemination to other tissues through interacting and degrading host hyaluronic acid. This would facilitate the movement of *T pallidum* through the extracellular matrix. Eventually the organism would reach vascular basement membrane and attach to it with ligands for collagen IV, fibronectin, or laminin, and would eventually penetrate the vessel. In immune rabbits the injection of organisms and local effects of treponemal hyaluronidase could induce rapid leakage of serum constituents, including antibodies, into the infected area. Anti-treponemal hyaluronidase could restrict movement through extracellular matrix, and then the antibodies could block attachment to basement membrane, thus restricting entry into the circulation.

G D Morrison

Ceftriaxone for treatment of primary syphilis in men: a preliminary study

T THIRUMOORTHY, C-T LEE, K-B LIM, T TAN (Singapore, Republic of Singapore). *Sex Transm Dis* 1987;**14**:116–8.

False positive results of tests for syphilis and outcome of pregnancy: a retrospective case-control study

JG THORNTON, GA FOOTE, CE PAGE, AD CLAYDEN, LAD TOVEY, JS SCOTT (Leeds, England). *Br Med J* 1987;**295**:355–6.

Gonorrhoea

Evidence of serum antibodies to *Neisseria gonorrhoeae* before gonococcal infection

CB HICKS, JW BOSLEGO, B BRANDT (Washington, USA). *J Infect Dis* 1987;**155**:1276–81.

Induction and repression of outer membrane proteins by anaerobic growth of *Neisseria gonorrhoeae*

VL CLARK, LA CAMPBELL, DA PALERMO, TM EVANS, KW KLIMPEL (Rochester, USA). *Infect Immun* 1987;**55**:1359–64.

Non-specific genital infection and related disorders (chlamydial infections)

A prospective study of *Chlamydia trachomatis* infection following legal abortion

G GIERTZ, I KALLINGS, M NORDENVALL, T FUCHS (Danderyd, Sweden). *Acta Obstet Gynecol Scand* 1987;**66**:107–9.

Unrecognised high prevalence of *Chlamydia trachomatis* cervical infection in an isolated Alaskan Eskimo population

KE TOOMEY, MP RAFFERTY, WE STAMM (San Francisco, USA). *JAMA* 1987;**258**:53–6.

Deoxyribonucleic acid hybridization analysis for the detection of urogenital *Chlamydia trachomatis* infections in women

CC PAO, S-S LIN, T-E YANG, Y-K SOONG, P-S LEE, J-Y LIN (Taipei, Republic of China). *Am J Obstet Gynecol* 1987;**156**:195–9.

In vitro evaluation of CP-62,993, erythromycin, clindamycin, and tetracycline against *Chlamydia trachomatis*

M WALSH, EW KAPPUS, TC QUINN (Baltimore, USA). *Antimicrob Agents Chemother* 1987;**31**:811–2.

In vitro activity of ciprofloxacin against *Chlamydia trachomatis*

J SCHACHTER, J MONCADA (San Francisco, USA). *Am J Med* 1987;**82** suppl 4A:42–3.

Treatment of *Chlamydia trachomatis* infections: comparison of 1– and 2–g doses of erythromycin daily for seven days
CC LINNEMANN, CL HEATON, M RITCHEY (Cincinnati, USA). *Sex Transm Dis* 1987;**14**:102–6.

Non-specific genital infection and related disorders (mycoplasmal and ureaplasmal infections)

Significance of antibodies to *Mycoplasma genitalium* in salpingitis
K LIND, GB KRISTENSEN (Copenhagen, Denmark). *Eur J Clin Microbiol* 1987;**6**:205–7.

Bacteraemia and pelvic infection in women due to *Ureaplasma urealyticum* and *Mycoplasma hominis*
DC PLUMMER, SM GARLAND, GL GILBERT (Fairfield, Australia). *Med J Australia* 1987;**146**:135–7.

Prevalence and significance of *Mycoplasma hominis* and *Ureaplasma urealyticum* in the urines of a non-venereal disease population
PM FURR, D TAYLOR-ROBINSON (Harrow, England). *Epidem Inf* 1987;**98**:353–9.

Suboptimal efficacy of erythromycin and tetracycline against vaginal *Ureaplasma urealyticum*
WR BOWIE, V WILLETTS (Vancouver, Canada). *Sex Transm Dis* 1987;**14**:88–91.

Non-specific genital infection and related disorders (general)

Etiology and manifestations of epididymitis in young men: correlations with sexual orientation
RE BERGER, D KESSLER, KK HOLMES (Seattle, USA). *J Infect Dis* 1987;**155**:1341–3.

Treatment of nongonococcal uethritis with ciprofloxacin
IW FONG, W LINTON, M SIMBUL, *ET AL* (Toronto, Canada). *Am J Med* 1987;**82** suppl 4A:311–6.

Pelvic inflammatory disease

Endometrial damage in acute salpingitis
ES TOMIOKA, RY ANZAI, WN KWANG (Sao Paulo, Brazil). *Sex Transm Dis* 1987; **14**:63–8.

Microbiological and histopathological findings in acute pelvic inflammatory disease
J PAAVONEN, K TEISALA, PK HEINONEN, *ET AL* (Tampere, Finland). *Br J Obstet Gynaecol* 1987;**94**:454–60.

The role of vaginal secretory immunoglobulin A, *Gardnerella vaginalis*, anaerobes, and *Chlamydia trachomatis* in postabortal pelvic inflammatory disease
L HEISTERBERG, PE BRANEBJERG, A BREMMELGAARD, J SCHEIBEL, L HØJ (Virum, Denmark). *Acta Obstet Gynecol Scand* 1987;**66**:99–102.

Trichomoniasis

Cell culture compared with broth for detection of *Trichomas vaginalis*
GE GARBER, L SIBAU, R MA, EM PROCTOR, CE SHAW, WR BOWIE (Vancouver, Canada). *J Clin Microbiol* 1987;**25**:1275–9.

The effects of inhibitors of sulphur-containing amino acid metabolism on the growth of *Trichomonas vaginalis in vitro*
K-W THONG, GH COOMBS (Glasgow, Scotland). *J Antimicrob Chemother* 1987;**19**:429–37.

Activities of metronidazole and miridazole against *Trichomonas vaginalis* clinical isolates
N YARLETT, H HOF, NC YARLETT (New York, USA). *J Antimicrob Chemother* 1987; **19**:767–70.

Genital herpes

Rapid diagnosis of genital herpes by detecting cells infected with virus in smears with fluorescent monoclonal antibodies
M BOTCHERBY, C GILCHRIST, J BREMNER, MA BYRNE, JRW HARRIS, D TAYLOR-ROBINSON (Harrow, England). *J Clin Pathol* 1987;**40**:687–9.

Treatment of first-attack genital herpes—acyclovir versus inosine pranobex
A MINDEL, G KINGHORN, E ALLASON-JONES, *ET AL* (London, England). *Lancet* 1987; i:1171–3.

Follow-up report on 50 subjects vaccinated against herpes genitalis with Skinner vaccine
GRB SKINNER, CG FINK, M COWAN, *ET AL* (Birmingham, England). *Med Microbiol Immunol (Berl)* 1987;**176**:161–8.

Genital warts

Detection of specific types of human papillomavirus in cervical scrapes, anal scrapes, and anogenital biopsies by DNA hybridization
BR HENDERSON, CH THOMPSON, BR ROSE, YE COSSART, BJ MORRIS (Sydney, Australia). *J Med Virol* 1987;**21**:381–93.

Cryotherapy for the treatment of proximal urethral condyloma acuminatum
PK SAND, W SHEN, LW BOWEN, DR OSTERGARD (Long Beach, USA). *J Urol* 1987;**137**:874–6.

Acquired immune deficiency syndrome

The psychiatry of HIV infection
SW BURTON (London, England). *Br Med J* 1987;**295**:228–9.

Pyomyositis associated with human immunodeficiency virus infection
RA WATTS, BI HOFFBRAND, DF PATON, JC DAVIS (London, England). *Br Med J* 1987; **294**:1524–5.

Necrotizing folliculitis in AIDS-related complex
RJ BARLOW, EJ SCHULZ (Pretoria, South Africa). *Br J Dermatol* 1987;**116**:581–4.

A case of malignant melanoma in AIDS-related complex
W KRAUSE, H MITTAG, U GIELER, E THOMAS, U WICHMANN (Marburg, Federal Republic of Germany). *Arch Dermatol* 1987;**123**:867–8.

Atopic manifestations in the acquired immune deficiency syndrome: response to recombinant interferon gamma
JM PARKIN, L-J EALES, AR GALAZKA, AJ PINCHING (London, England). *Br Med J* 1987;**294**:1185–6.

Chronic vaginal candidiasis in women with human immunodeficiency virus infection
JL RHOADS, DC WRIGHT, RR REDFIELD, DS BURKE (Washington, USA). *JAMA* 1987;**257**:3105–7.

Nonspecific interstitial pneumonitis: a common cause of pulmonary disease in the acquired immunodeficiency syndrome
AF SUFFREDINI, FP OGNIBENE, EE LACK (Bethesda, USA). *Ann Intern Med* 1987;**107**:7–13.

Cardiorespiratory arrest and autonomic neuropathy in AIDS
C CRADDOCK, G PASVOL, R BULL, A PROTHEROE, J HOPKIN (Oxford, England). *Lancet* 1987;ii:16–8.

Obstetric and perinatal consequences of human immunodeficiency virus (HIV) infection: a review
CS PECKHAM, YD SENTURIA, AE ADES (London, England). *Br J Obstet Gynaecol* 1987; **94**:403–7.

Acquired immune deficiency syndrome presenting as a progressive infantile encephalopathy
SL DAVIS, CC HALSTED, N LEVY, W ELLIS (Sacramento, USA). *J Ped* 1987;**110**:884–8.

Infants born to mothers seropositive for human immunodeficiency virus: preliminary findings from a multicentre European study
JQ MOK, C GIAQUINTO, A de ROSSI, I GROSCHWÖRNER, AE ADES, CS PECKHAM (London, England). *Lancet* 1987;i:1164–8.

Human-immunodeficiency-virus infections in infants negative for anti-HIV by enzyme-linked immunoassay
W BORKOWSKY, K KRASINSKI, D PAUL, T MOORE, D BEBENROTH, S CHANDWANI (New York, USA). *Lancet* 1987;i:1168–71.

Isolation of human immunodeficiency virus and detection of HIV DNA sequences in the brain of an ELISA antibody-negative child with acquired immune deficiency syndrome and progressive encephalopathy
MV RAGNI, AH URBACH, S TAYLOR, *ET AL* (Pittsburgh, USA). *J Ped* 1987;**110**:892–4.

Transmission of human immunodeficiency virus from parents to only one dizygotic twin
CL PARK, H STREICHER, R ROTHBERG (Chicago, USA). *J Clin Microbiol* 1987;**25**:1119–21.

Non-cytocidal natural variants of human immunodeficiency virus isolated from AIDS patients with neurological disorders
R ANAND, F SIEGAL, C REED, T CHEUNG, S FORLENZA, J MOORE (Washington, USA). *Lancet* 1987;ii:234–8.

Butylated hydroxytoluene, lipid-enveloped viruses, and AIDS
E REIMUND (Little Rock, USA). *Medical Hypotheses* 1987;**23**:39–42.

The San Francisco men's health study: III. Reduction in human immunodeficiency virus transmission among homosexual/bisexual men, 1982–86
W WINKELSTEIN, M SAMUEL, NS PADIAN, *ET AL* (Berkeley, USA). *Am J Public Health* 1987;**77**:685–9.

Human immunodeficiency virus infections among civilian applicants for United States military service, October 1985 to March 1986
DS BURKE, JF BRUNDAGE, JR HERBOLD, *ET AL* (Washington, USA). *N Engl J Med* 1987;**317**:131–6.

Retrospective seroepidemiology of AIDS virus infection in Nairobi populations
P PIOT, FA PLUMMER, M-A REY, *ET AL* (Antwerp, Belgium). *J Infect Dis* 1987; **155**:1108–12.

Human T-lymphotropic virus type 4 and the human immunodeficiency virus in West Africa
PJ KANKI, S M'BOUP, D RICARD, *ET AL* (Boston, USA). *Science* 1987;**236**:827–31.

Risk of AIDS after herpes zoster
M MELBYE, RJ GROSSMAN, JJ GOEDERT, ME EYSTER, RJ BIGGAR (Aarhus, Denmark). *Lancet* 1987;i:728–31.

A longitudinal clinical study was performed on about 2800 homosexual men attending a private clinic in New York. Herpes zoster developed in 112 men. Although three of the men already had the acquired immune deficiency syndrome (AIDS), herpes zoster antedated this condition in 24 of the remaining 109 men. Using Kaplan-Meier survival analysis, the estimated proportions of men with herpes zoster who would subsequently develop AIDS were 22·8% after two years, 45·5% after four years, and 72·8% after six years. The risk of AIDS developing was appreciably increased in patients with herpes zoster who presented with severe clinical disease, disease affecting the neck, or severe pain. Associated oral candidiasis, oral hairy leukoplakia, amoebiasis, or superficial fungal infections also indicated an increased risk of AIDS. Factors with no predictive value included multiple dermatome herpes zoster and persistent glandular lymphadenopathy.

The value of the paper is diminished slightly because the overall HIV status of the study population was not known, but was predicted from a cohort of 86 homosexual men from the same practice. Current multicentre follow up studies of HIV positive patients may confirm herpes zoster as being one of many clinical correlates of the development of AIDS. In the future, clinical and immunological criteria will identify asymptomatic men who may be considered for early treatment before end stage disease develops.

E Monteiro

Predictors of clinical AIDS in young homosexual men in a high-risk area
R DETELS, B VISSCHER, JL FAHEY, *ET AL* (Los Angeles, USA). *Int J Epidemiol* 1987;**16**: 271–6.

The prevalent cohort study and the acquired immunodeficiency syndrome
R BROOKMEYER, MH GAIL, BF POLK (Baltimore, USA). *Am J Epidemiol* 1987;**126**:14–24.

Serologic and immunologic studies in patients with AIDS in North America and Africa: the potential role of infectious agents as cofactors in human immunodeficiency virus infection
TC QUINN, P PIOT, JB McCORMICK, *ET AL* (Baltimore, USA). *JAMA* 1987;**257**:2617–21.

Serological and immunological studies were performed on 38 African (20 men, 18 women) and 60 homosexual American patients with the acquired immune deficiency syndrome (AIDS). Thirty six (95%) of the 38 African patients with AIDS (all of whom were under 60 and had evidence of either opportunistic infection or disseminated Kaposi's sarcoma) and 58 (97%) of the American patients with AIDS (Centres for Disease Control Surveillance definition) were seropositive to the human immunodeficiency virus (HIV) using a commercially available ELISA and by western blot analysis.

One hundred age and sex matched African outpatient men and women, 100 homosexually active American men, and 100 American heterosexual outpatient men were selected randomly as controls. None of the controls had evidence of either opportunistic infection or Kaposi's sarcoma at the time of enrolment. None of the American heterosexual men were human immunodeficiency virus (HIV) antibody positive compared with 22 of the 100 homosexual American men and six of the 100 African controls.

Common to African patients with AIDS, African outpatient controls, American patients with AIDS, and homosexual men was the finding of extremely high prevalences of antibody to cytomegalovirus, hepatitis B virus, hepatitis A virus, syphilis, *Toxoplasma gondii*, or Epstein-Barr capsid antigen. In contrast, the prevalence of antibody to each of these infectious agents was significantly lower among the 100 American heterosexual men ($p < 0·001$).

When comparing immunological features in each of the three control populations, patients with HIV infection were excluded from the analysis, which subsequently enabled the authors to draw several important

immunological comparisons. Firstly, American heterosexual men were found to have a significantly higher cell population of T helper cells compared with the African outpatient and American homosexual groups ($p < 0.01$). Secondly, the absolute numbers of activated T lymphocytes were lower in the American heterosexual men than in all four other groups. Thirdly, the African outpatient controls had significantly more absolute numbers of activated T lymphocytes than the other patient populations ($p < 0.001$). Finally, African outpatient controls had higher levels of immune complexes than American controls.

This study provides us with evidence of immunological alterations in an African population uninfected with HIV, and similarly the authors propose that common exposure to multiple microbial agents, not only the infections documented but also malaria, trypanosomiasis, and filariasis (all of which are known to have a major effect on the immune system) could act collectively or individually to render a person more susceptible to HIV infection. It is not unreasonable to speculate, for example, that the increased circulating activated T lymphocytes noted in the African controls would readily support productive infection with HIV. These findings, with other epidemiological factors including heterosexual promiscuity, exposure to infected blood transfusions and to blood contaminated needles, and high rates of untreated venereal disease associated with open genital lesions, may help to explain the rapid spread of HIV infection in the general heterosexual population of African countries.

W C Stack

Association of different allelic forms of group specific component with susceptibility to and clinical manifestation of human immunodeficiency virus infection

L-J EALES, KE NYE, JM PARKIN, *ET AL* (London, England). *Lancet* 1987;i:999–1002.

The authors studied the allelic incidence and distribution of phenotypes of the group specific component (Gc) in control groups of heterosexual and homosexual men compared with homosexual men at risk of human immunodeficiency virus (HIV) infection and homosexual men with HIV infection. Gc is an α 2 globulin found on cells and in serum. There are three alleles Gc 1 fast (Gc 1f), Gc 1 slow (Gc 1s), and Gc 2; the phenotype is determined by mendelian inheritance and may be homozygous (for instance, Gc 1f 1f) or heterozygous (for instance, Gc 1f 1s).

Compared with the control group of HIV negative heterosexuals, seronegative contacts of HIV showed significantly more phenotype Gc 2 2 and less Gc 1f 1f. In contrast the HIV positive groups showed less Gc 2 2, but significantly more Gc 1f 1f. The χ^2 test for association was used to assess the correlation between Gc, susceptibility or resistance to HIV infection, and the severity of clinical symptoms. Increasing severity of clinical symptoms showed a positive association with the Gc 1f allele but negative associations with Gc 2 and Gc 1s. Once divided into the phenotypes the numbers in the study groups were small, particularly for the statistical methods used. It was, however, striking that no patient with AIDS was phenotype Gc 2 2, and no HIV negative high risk patient was Gc 1f 1f.

The authors concluded that in homosexual men Gc phenotypes influence susceptibility to HIV infection, and in people infected with HIV Gc phenotypes may influence the severity of illness. They also noted that in parts of central Africa the Gc 1f allele predominates. They suggested a possible mechanism for this. The alleles differ biochemically by a small number of peptides; Gc 1f contains two sialic acid residues, Gc 1s has one residue, and Gc 2 none. They suggest that HIV may bind these sialic acid residues on Gc, thus facilitating HIV entry into host cells. Gc 1f would thus increase susceptibility to HIV infection and spread from cell to cell in an infected person, thereby affecting the rate of progression of disease.

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